Correspondence

Magico-religious Mercury Exposure

Mark Wheeler's Focus article, "Measuring Mercury" (*1*), which appeared in the August 1996 issue of EHP, contained a serious omission. Wheeler concentrated on methyl mercury and, to a lesser extent, elemental mercury in dental amalgams. He failed to mention the relatively recently described but extremely significant exposures to elemental mercury in ethnically Hispanic and Caribbean homes, consequent to its use for a variety of magico-religious and ethnomedical purposes (*2–3*).

Such domestic use and presumed exposure has been documented in a number of published papers, as well as by research sponsored by the ATSDR (*4–6*) and the EPA (*7*). In fact, an ATSDR monograph specifically alerts clinicians to this exposure pathway: "Metallic mercury has been used by Mexican-Americans and Asian populations in folk remedies for chronic stomach disorders and by Latin-American and Caribbean natives in occult practices" (*4*). This monograph was edited by Thomas Clarkson, who was interviewed by Wheeler, and who has long been aware of elemental mercury's domestic use. Similarly, the EPA's Kathryn Mahaffey, also interviewed, has been aware of domestic mercury exposure for some years, and the EPA issued a risk assessment document on cultural uses of mercury in 1993 (*7*).

These mercury exposures are especially significant from an environmental health perspective because, in many cases, they are certain to be orders of magnitude greater than (methyl) mercury exposures from eating fish or from the leaching of mercury in amalgam fillings. Additionally, the mercury vapor released from mercury intentionally sprinkled on floors affects all occupants of contaminated homes, from the fetus to the elderly.

Andrew Rowland, cited in "The Issue of Amalgams" (*1*), has been aware of domestic mercury exposure for several years. Rowland makes a call for more research on health effects of amalgam–mercury exposure. I make a similar call for research on magico-religious mercury exposure. If the environmental health research community continues to ignore magico-religious mercury exposure, its health effects will never be ascertained.

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MMA:DMA Ratios Reversed

I would like to bring to your attention an apparent typo in a recent response written by Mushak and Crocetti in Environmental Health Perspectives (*1*). In describing a publication by Warner et al. (*2*), they note that "the corresponding MMA:DMA ratios for exposed and control subjects were 0.32 and 0.5 ..." (p. 1017, first column). These values should be reversed.

As reported by Warner et al. (*2*) and correctly cited by Mushak and Crocetti in their original commentary (*3*), urinary arsenic concentrations were 190 mg MMA/l and 390 mg DMA/l for the exposed group, and 14 mg MMA/l and 44 mg DMA/l for the control group. Therefore, the actual MMA:DMA ratios should be 0.49 (190/390) for the exposed group and 0.32 (14/44) for the control group. These correct ratios are consistent with our hypothesis that MMA:DMA ratios tend to be higher in exposed populations and that methylation may be less efficient as dose increases. For clarification, this error should be noted.

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Response

Dr. Slayton calls attention to a minor typo in our response letter to Slayton et al. in the October 1996 EHP (*1*). She notes that the two ratios of MMA:DMA were in reverse order relative to the antecedent corresponding. The fact of the typo is correct as the wording appeared and you may wish to note a correction. The consequence of the typo, however, is nil for any of our interpretations in the response and the original commentary and therefore requires no editorial amplification by EHP.

As we noted in the commentary (*2*) and in the response to Slayton et al. (*1*), the Nevada MMA:DMA ratios of methylated arsenic compared to a control group are relatively insignificant as to change despite the high water arsenic exposures. This was and remains the main point. The amount of change is equally modest whether one is comparing 0.32 to 0.50 or 0.50 to 0.32. The context makes it clear what was intended. Getting these values reversed has no impact on anything we said or have interpreted in either article. Dr. Slayton seems to think the fact of the reversal of ratios would likewise compel us to reverse our conclusions and magnify the arguments of Slayton et al. That is not the case. We knew what was intended in both the commentary (*2*) and the letter (*1*).

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Reply to Comments on "A Reevaluation of Cancer Incidence Near the Three Mile Island"

In their letter (*1*), Hatch et al. appear to be confused about the purpose of Table 1 of our paper (*2*); the table merely shows that the differences between our results and theirs are not due to statistical methods. Our Tables 2 and 3 show that underascertainment of incident cancers, data management errors, and failure to adjust for baseline variation in cancer rates led Hatch et al. (*3*) to underestimate associations between estimated radiation doses from

the TMI accident and cancer incidence in the 10-mile area around the plant. Contrary to Hatch et al.'s assumption that undercounted cancers in 1975 would not bias associations (*1*), the incomplete ascertainment of cases led them to underestimate the dose–effect association, in particular for lung cancer.

However, logical, not technical, errors were the focus of our paper. Hatch et al. specified childhood cancer as a primary outcome (*3*) but failed to consider birth cohorts. Consequently, their analysis counted among the exposed many children who were not conceived at the time of the accident. Our analyses focused on broad categories of cancer that might be expected to be related to population exposure to radiation and that occurred at a sufficient frequency to support dose–response analyses.

Most importantly, Hatch et al.'s study (*3*) appeared constrained by circular reasoning. Hatch et al. assumed that the maximum radiation dose was "very low, an average of approximately 0.1 mSv, with 1 mSv the projected maximal dose" (*3*). Based on National Academy of Sciences risk estimates (*4*), a dose of 1 mSv would result in an increase in cancer of less than a half percent (i.e., a relative risk or observed/expected ratio of 1.005). Consequently, after observing that cancer rates rose with estimated accident doses, they concluded that this association might be evidence of stress among the exposed, rather than the effect of radiation from the TMI accident (*5*), which was their primary hypothesis. In contrast, we considered the possibility that exposures for some populations may have been substantially higher than the 1 mSv assumed by Hatch et al. (*3*) and that evidence of an observed association could lead to rejection of the null hypothesis of no accident effect on cancer incidence.

Hatch et al. should be familiar with reasons for questioning the assumption that the maximum dose received from the TMI accident was 1 mSV. Contrary to their statement that cytogenetic damage was the "sole supporting evidence" that led us to consider the possibility of high doses (*1*), the document they cited (*6*) in their article (*3*), as well as proceedings from the TMI Public Health Fund (*7*), report conditions that would be expected following high radiation doses, including hair loss, vomiting, nausea, animal deaths, and excess cancers. Further, models used to estimate exposure were based on inadequate data because of inoperable radiation monitors, sparse placement of dosimeters, and unavailability of detailed meteorological data for the study areas. Radiation monitoring data were particularly inadquate for releases that occurred early in the accident.

Hatch et al.'s contention that "there were no court-imposed limitations on our exposure models" seems to conflict with their statement that "the only limitation involved our agreement to use an exposure model rather than upper limit dose calculations, which are not suitable for an epidemiological study in the first place" (*1*). They do not note that the court order did permit them to assume "upper limit or worst case estimates of releases of radioactivity" if this scenario resulted in an estimate of "less than 0.01 health effects," and they fail to mention that the order further required approval of their findings by nuclear industry attorneys (*8*). Under most circumstances, a worst case scenario would appear to be an unreasonable assumption for a scientific study in which best estimates would be used rather than extremes in either direction. However, there is extensive evidence that the industry and government agencies responsible for the operation of the TMI plant, reporting of releases, and protection of the public, have a long history of showing greater concern for public image than for full disclosure of occupational and environmental radiation exposures and evidence of radiation health effects (*9–13*).

Hatch et al. (*1*) write that they "considered but rejected a post- versus pre-accident analysis." This statement conflicts with the Methods section of their paper in which such an approach is described and with a result (for lung cancer) that they say was based on that strategy (*3*). Their argument that the plant was operational in the pre-accident period has no bearing on this analytical strategy, as concern about routine emissions would only provide further rationale for considering adjustment for baseline differences in cancer rates.

The most puzzling comment in Hatch et al.'s letter (*1*) is that "Wing et al. do not seem to have adjusted their standard errors to reflect the *a posteriori* nature of their hypotheses." We addressed the very same hypothesis that Hatch et al. investigated, but without the assumption that results of the analysis could not support the hypothesis. However, even if we had addressed a different hypothesis, we do not believe that adjustment of standard errors would be appropriate, given that the study was based on a complete population, not a random sample of a population to which an inference was being made. In such a situation, standard errors and tests of goodness of fit are more appropriately treated as important descriptive information about the precision of the effect estimates rather than in terms of random sampling from a fixed population (*14*).

Although the total population size in the TMI 10-mile area was given in our paper (*2*), and the size of populations in the four dose categories created by Hatch et al. were given in their paper (*3*), we did not include the population size in each of the nine dose groups shown in our Table 3 (*2*). This information would be helpful for interpreting the population impact of the accident, and we give those numbers here. The average population size during the 1981–1985 post-accident time period in each dose group from the lowest to the highest was 6,683; 36,088; 11,368; 32,606; 15,518; 33,794; 6,852; 12,536; and 7,356.

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Erratum

In the Correspondence published by Mushak and Crocetti (Response: Accuracy, Arsenic, and Cancer) published in EHP in Volume 104, Number 10, 1996, one sentence was printed incorrectly. The correct sentence is as follows: "the corresponding MMA:DMA ratios for exposed and control subjects were 0.50 and 0.32 ..."